▶15 Headache

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Importance

Headache has a high incidence and prevalence in the community, with associated morbidity. The prevalence of migraine in the last year was 6% of males and 15% of females in a Danish epidemiological study where a random sample of 25 to 64-year-olds was examined by neurologists.[1] These prevalence figures are consistent with other population studies using the International Headache Society (IHS) criteria.[2,3] The prevalence of tension headache in the last year in the Danish study was 63% among males and 86% among females.[4] Daily or near-daily headache affects almost 5% of unselected populations,[5,6] of which half is chronic tension-type headache.

Headache is commonly presented in primary care. The Fourth National Morbidity Study found a consultation rate for migraine of 115/10 000 person years at risk;[7] an incidence study in a British general practice population where new cases over 1 year were noted found an incidence of 210/100 000 for new headache and 64/100 000 for new migraine.[8]

Review of evidence relating to quality indicators

Diagnosis

Headaches are largely diagnosed as clinical syndromes. Primary headaches are those for which there is no structural or metabolic cause. The objective of the diagnostic process is to rule out secondary causes, particularly serious causes of headache, and then to accurately diagnose primary headache to ensure appropriate treatment.

History

The most common primary headaches are tension headache and migraine. Secondary headache is most commonly associated with excess alcohol consumption, medication misuse, fever and disorders of the nose and sinuses.[3] Headache type is diagnosed by focusing on the exact definition of the prominent symptoms, in particular the temporal pattern, the description of pain and associated signs and symptoms, as well as related phenomena. Primary headaches can coexist: a patient may have tension headache and migraine.

The headache history should include whether this is new-onset headache or part of a longer headache history and if so, the age of onset of headache. Characteristics of the

headache itself need to be assessed, such as the location (migraine is usually unilateral but 30–40% can be bilateral), the quality (e.g. pulsating, pressure), the intensity/severity and any aggravation by movement. Prodromal features or aura are clinical features of classical migraine. Nausea or vomiting during an attack, and sensitivity to light and sound predominantly occur with migraine. Other features of headache history include family history, precipitating, exacerbating or palliating factors and a drug history.

Aspects of the history that may indicate underlying pathology are new-onset headache, particularly in middle age or later, worst-ever headache, changes in frequency or severity of usual features of previous long-standing headache, and the presence of systemic features such as fever, anorexia, weight loss, muscle ache or jaw claudication.[9]

There is very little literature on the sensitivity and specificity of aspects of the history. In a Danish population, a questionnaire validated against a telephone interview found that the two questions 'Have you ever had migraine?' and 'Have you ever had visual disturbances lasting 5–60 min followed by headache?' identified 93% of migraineurs with aura and 75% of migraineurs without aura.[10] The findings are likely to differ in populations with less knowledge of migraine. Jaw claudication is strongly suggestive of giant cell arteritis; the odds of a positive biopsy are 9 times greater if jaw claudication is present.[11] Individual items of the clinical history have low accuracy for the diagnosis of meningitis in adults. Results from a review of studies of patients with meningitis indicated sensitivity of 50% for headache (CI 32–68%) and sensitivity of 30% for nausea/vomiting (CI 22–38%).[12]

There is evidence of comorbidity of headache and mood disorders.[13–17] These areas may need to be covered in the history.

Examination

There is lack of agreement about the essential elements of the physical examination. If the main symptom is headache, and the history has not revealed any focal symptoms, complete physical and neurological examination is not usually helpful.[18] Examinations suggested by neurologists include blood pressure and extracranial structures such as sinuses, scalp arteries, cervical paraspinal muscles and temporomandibular joints (TMJs). Suggested neurological examination includes optic fundi, cranial nerves (in particular the Vth cranial nerve, including the corneal reflex); muscle power, gait, reflexes and plantar responses. The prime objective of the examination is to support the diagnostic hypothesis suggested by the history and to exclude causes of headache that require further investigation.

There is a lack of evidence as to the precise value of most elements of the examination, and no evidence as to their predictive value in a general practice population. A questionnaire designed in an American headache centre to screen for temporomandibular disorders found high sensitivity and specificity (92% and 91% respectively) for pain with maximum jaw opening using passive stretch and reciprocal jaw clicking or pain on palpitation over TMJ, to distinguish TMJ derangement from headache patients.[19] One case control study in a headache centre found that papilloedema was an uncommon sign in acute elevation of intracranial pressure.[20]

Investigation

If a headache conforms to the diagnostic criteria of the IHS, there is little value in imaging techniques such as CT scan or MRI. Frishberg[21] reviewed the literature concerning the putative associations between various headache types and intracranial pathology, and the literature pertaining to neuroimaging. He concluded that intracranial pathology was rare in patients whose headaches fitted IHS or other accepted criteria. In patients thought to have migraine he recommended imaging as follows: in patients with onset after the age of 60, mental or personality changes, bruits, focal neurology, history of seizures, or those with hemiplegic, basilar or ophthalmoplegic migraine. In cases with onset after the age of 40, no family history, change in pattern, prolonged aura, side-locked headache and resistance to treatment imaging should be considered, but these cases are likely to have very low yield. In the case of headaches other than migraine or cluster headache, he concluded that the literature does not provide enough data to make statistically predictive observations in patients with headache but no findings on neurological examination.

Laboratory investigations such as Hb, CRP and ESR are suggested when the diagnosis of temporal arteritis is suspected. However, evidence from the literature is conflicting. One retrospective study of patients referred for biopsy found no significant difference in either parameter in biopsy-positive or biopsy-negative cases.[22] However, a study using controls as well as the group referred for biopsy found a sensitivity of 100% for CRP, 92% for ESR and the combination of ESR and CRP was highly specific (97%) for temporal arteritis.[11]

Referral (indicator 1)

The Referral Guidelines for Suspected Brain Tumour suggest that a referral should be made and patient seen within 2 weeks for:

- subacute progressive focal neurological deficit (e.g. weakness, sensory loss, dysphasia, ataxia)
- new-onset seizures characterised by one or more of
  - focal seizures
  - prolonged (greater than 1 hour) post-ictal deficit
  - status epilepticus
  - associated interictal focal deficit
- patients with headache, vomiting and papilloedema,
- cranial nerve palsy (e.g diplopia, visual failure including optician-defined visual field loss, unilateral sensorineural deafness).

Urgent referral should be considered for

- headache patients with non-migrainous headaches of recent onset, present for at least 1 month, when accompanied by features suggestive of raised intracranial pressure (e.g woken by headache, vomiting, drowsiness)

Treatment

This section deals with the evidence for the treatment of primary headache. We have confined our comments to therapeutic agents likely to be used in a UK primary care context. Experts comment on the importance of eliminating triggers, that stress can precipitate any type of headache and that non-drug treatment such as relaxation should be considered.

In the case of migraine, treatment options vary as to the severity of the acute attack. Limmroth et al.[23] reviewed the use of aspirin (acetylsalicylic acid or ASA) in acute treatment and prophylactic treatment of migraine. In the acute phase its effectiveness is enhanced by an effervescent form and combination with antiemetics (of note perhaps is that the dose of ASA used in studies had been of the order of 900 mg). In a multicentre trial, water-soluble aspirin with metoclopramide was as effective as 100 mg of oral sumatriptan in reducing headache intensity and autonomic features such as nausea, and it had fewer adverse effects.[24] Ibuprofen, naproxen and diclofenac sodium have been shown to be effective treatments for an acute attack.[25–27] 50 mg of oral diclofenac K provided more rapid relief than oral sumatriptan in a double-blind randomised crossover trial.[28] (The potassium salt is more rapidly absorbed than the sodium.)

Triptan use is recommended for moderate or severe migraine. A systematic review found that subcutaneous sumatriptan was more efficacious than the oral form and had a quicker onset of action, but caused more side-effects.[29] The investigator stated that the majority of side-effects were minor and may be tolerable to patients. A prospective study of the tolerability of subcutaneous sumatriptan in acute migraine, which followed 12 239 patients over 12 months, found no major adverse effects when sumatriptan was used according to the precautions and warnings on the label.[30]

There is evidence from randomised controlled trials for the effectiveness of intramuscular chlorpromazine, metoclopramide and prochlorperazine for an acute attack.[31,32]

Overuse of most acute attack therapies can aggravate headache frequency, and particular care needs to be taken with codeine-containing compound analgesics.

Prophylactic treatment is suggested if attacks occur frequently, or if attacks severely incapacitate the patient (indicator 2). Traditionally, experts have suggested that prophylaxis should be considered if a patient suffers 2 attacks or more each month, but more effective remedies for the acute attack may vary this. The efficacy of commonly used prophylactic drugs is low.[33]

There is clinical trial evidence for the effectiveness of the following drugs in prophylaxis (only propanolol and pitozifen are licensed for migraine prophylaxis in the UK): beta-blockers (atenolol, metoprolol, naldolol, propanolol);[34–36] calcium channel blockers (verapamil, flunarizine);[37,38] serotonin receptor antagonists (pizotifen, methysergide);[39,40] tricyclics (amitriptyline);[41] and 'antiepileptics' (sodium valproate, valproaic acid)[42,43] (indicator 3).

There is evidence for the effectiveness of naproxen but the expert recommendation is to confine its use to the prophylaxis of perimenstrual migraine as intermittent use results in fewer gastrointestinal side-effects.[44,45] Expert opinion is that failure of one

beta-blocker does not necessarily predict the response to another. Treatment should start with a low dose and titrate upwards; once pain is controlled, the dose may be tapered down.

In the treatment of headache other than migraine, aspirin and ibuprofen have been shown to be effective in the treatment of episodic tension-type headache.[46] Dosage may be important: a double-blind placebo-controlled comparison of ketoprofen 25 mg and paracetamol 1000 mg found that both were more effective than placebo, with no significant difference between the active agents;[47] and a comparison of ibuprofen 400 mg with paracetamol 1000 mg found ibuprofen to be the more effective.[48] Tricyclics have been found to be effective in reducing the frequency and severity of chronic tension-type headaches.[49]

There is randomised controlled trial evidence for the effect of spinal manipulation in the case of cervicogenic headache.[50] However, spinal manipulation does not have a positive effect on episodic tension-type headache.[51]

Certain prophylactic agents should not be prescribed in specific at-risk groups, for example sumatriptan for patients with angina and beta-blockers for asthmatics (indicator 4).

Follow-up

Experts recommend the use of headache diaries in the assessment and treatment of headache.[9,52] Prophylactic treatment also requires regular monitoring.

Headache in children and adolescents

The diagnosis and treatment of headache in children is broadly similar to that in adults. The criteria for the diagnosis of migraine in children is different, in part relating to aspects of the headache itself, but also noting the difficulty that children may have in articulating characteristics such as the intensity and the nature of pain.[53] Mortimer et al.[54] interviewed children registered at one general practice. Headache prevalence increased from the age of 3 years up to age 11 in boys and girls, with a higher prevalence in 3 to 5-year-old boys than 3 to 5-year-old girls. The overall prevalence of headache was 38.8%, and of migraine was 3.7%. There is a suggestion that tension-type headache in children is associated with psychosocial problems, but the evidence from the literature is conflicting.[55] The history and examination should be supplemented by a developmental assessment. Simple analgesia such as paracetamol is suggested, used early and in adequate dosage (Committee on Safety of Medicines advice is to avoid the use of aspirin in children under 12 years, except for specific indications, due to the risk of Reye's syndrome). Metoclopramide can cause severe extrapyramidal side-effects in children. Zolmitriptan is licensed for use from the age of 12. Pizotofen is used for prophylaxis.

Recommended quality indicators for headache

Diagnosis/referral

1. Patients should be referred urgently for specialist care and investigation if the presenting headache is accompanied by:
   a. suspected raised intracranial pressure
   b. new-onset seizure
   c. focal neurological signs
   d. papilloedema

Treatment

2. Prophylaxis treatment should be offered in patients with severe and disabling migraine
3. The following agents should be prescribed as first line for prophylaxis of migraine unless contraindicated:
   a. beta-blocker
   b. tricyclic antidepressant
   c. pizotifen
4. a. Sumatriptan should not be prescribed for migraine in patients with angina
   b. Beta-blockers should not be prescribed for migraine in patients with asthma

Overview of data sources used in this review

Articles on headache were identified by a search of the 1991–5 and 1996–9 Medline databases and the Cochrane library. Keywords in the search were 'headache' (exploded and used as textword), 'family practice', 'epidemiology', 'sensitivity and specificity', and 'treatment'. We searched particularly for literature with a community/general practice focus. However, the majority of evidence is from specialist centres and uses the classification of headache of The International Headache Society (IHS).[56] This establishes diagnostic criteria for headache using a hierarchical scale and has been used since 1988 for epidemiological and clinical research.[56] Guidelines for the diagnosis and management of headache and migraine have been produced by a number of groups and these were also reviewed.[57,58] Migraine management guidelines have been produced by the British Association for the Study of Headache[59] and a group endorsed by Migraine in Primary Care Advisors.[60] Referral Guidelines for Suspected Brain Tumours have been prepared as part of the initiative to allow patients with suspected cancer to be seen by a specialist within 2 weeks.

There is an absence of evidence relating to the management of headache in primary care practice and many aspects of diagnosis and treatment are based on expert opinion.

Further reading

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Pryse-Phillips WE, Dodick DW, Edmeads JC et al. Guidelines for the diagnosis and management of migraine in clinical practice. *Canadian Medical Association Journal* 1997; 156: 1273–1287

The British Association for the Study of Headache (BASH) has a website at www.bash.org.uk

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Upper respiratory tract infections

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Importance

Acute upper respiratory tract infections (URTIs) are the commonest reason for patients to seek medical advice[1] and also the commonest reason for antibiotics to be prescribed. The current major concern is that the inappropriate use of antibiotics for usually self-limiting conditions will foster the development of antibiotic resistance and lead to serious infections becoming untreatable.[2–6] For this reason it is currently a national priority not to encourage the use of antibiotics unless there is very good evidence of their efficacy.

Pharyngitis/tonsillitis: diagnosis

Pharyngitis is caused by both bacterial and viral organisms.[7] Antibiotics could be targeted to those who have positive throat swabs for group A Streptococcus, a positive rapid Strep. test, or clinical characteristics associated with a positive throat swab.[8–11] Alternatively throat swabs could be used in selected populations.[11] However, throat swabs increase costs significantly, potentially medicalise self-limiting illness[12] and rarely modify clinical decisions.[13] In addition, antibiotic use and overall accuracy of decision making may be unchanged.[14] The throat swab is neither particularly sensitive nor specific when compared to a rise in antistreptolysin-O titres (ASOT). This is the case in both the general population[15] and in selected general practice populations where clinical selection has occurred.[16] Clinical scores or decision rules based on the throat swab[8–11] have the same limitations of validity as the throat swab, although they may crudely identify patients at risk of complications (see below).

Pharyngitis/tonsillitis: treatment

Antibiotics

A systematic review[17] indicates that antibiotics reduce symptom duration by a few hours to half a day. For patients who are not systemically unwell, either not prescribing or using a delayed prescribing approach (waiting for several days before using the prescription) is acceptable, changes attitudes to antibiotics, modifies attendance behaviour and does not delay symptom resolution appreciably.[18,19] Delaying the prescription probably results in 20% fewer recurrences compared to the immediate prescriptions of antibiotics, presumably because antibiotics modify local or systemic immune mechanisms.[20–22]